PB87-102802

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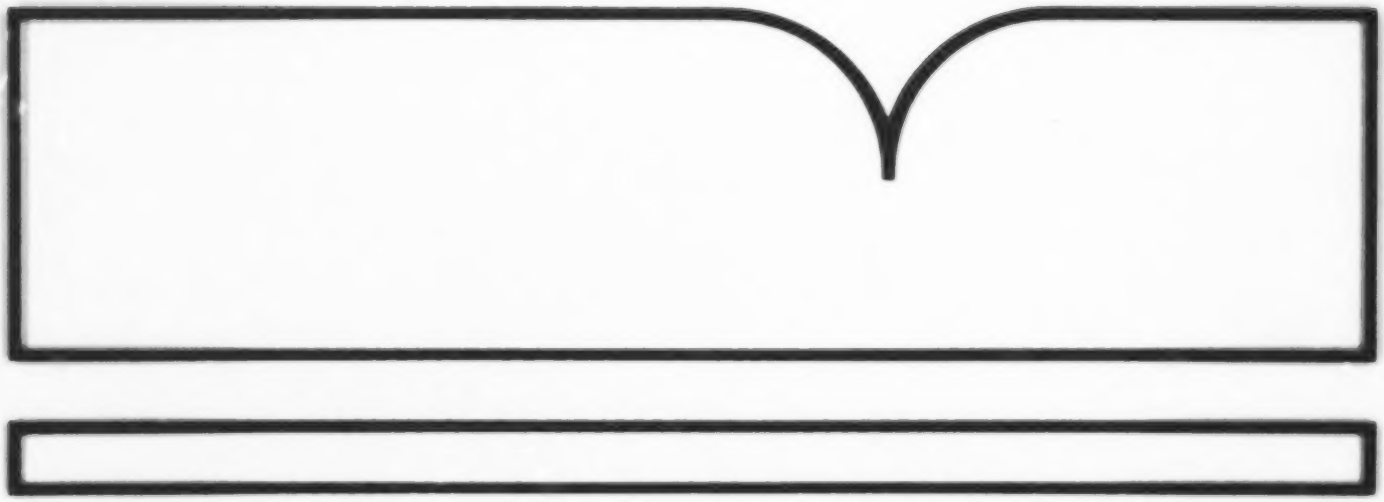
(U.S.) Health Effects Research Lab.
Cincinnati, OH

Sep 86



U.S. Department of Commerce
National Technical Information Service
NTIS

PB87-102802

EPA/600/D-86/212
September 1986

DRINKING WATER TRANSMISSION OF GIARDIASIS IN THE UNITED STATES

by

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TECHNICAL REPORT DATA
(Please read Instructions on the reverse before completing)

1. REPORT NO.	2.	3. RECIPIENT'S ACCESSION NO.
EPA/600/D-86/212		

4. TITLE AND SUBTITLE	5. REPORT DATE
DRINKING WATER TRANSMISSION OF GIARDIASIS IN THE UNITED STATES	September 1986
	6. PERFORMING ORGANIZATION CODE

7. AUTHOR(S)	8. PERFORMING ORGANIZATION REPORT NO.
E. W. Akin and W. Jakubowski	

9. PERFORMING ORGANIZATION NAME AND ADDRESS	10. PROGRAM ELEMENT NO.
Health Effects Research Laboratory (HERL)-RTP, NC Office of Research and Development (ORD) U.S. Environmental Protection Agency (USEPA) Research Triangle Park, North Carolina 27711	ABMC1A 11. CONTRACT/GRANT NO.

12. SPONSORING AGENCY NAME AND ADDRESS	13. TYPE OF REPORT AND PERIOD COVERED
Health Effects Research Laboratory (HERL)-RTP, NC Office of Research and Development (ORD) U.S. Environmental Protection Agency (USEPA) Research Triangle Park, North Carolina 27711	Published Paper 14. SPONSORING AGENCY CODE EPA/600/11

15. SUPPLEMENTARY NOTES

P.O. Elmer W. Akin, TMD/HERL/ORD/USEPA (CI) - Presented at 13th Biennial International Conference on Water Pollution & Control; Rio de Janeiro, Brazil; August 17-23, 1986

16. ABSTRACT

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Further evidence for waterborne transmission of Giardia came in 1975 with the first isolation of the organism from a water supply implicated in an outbreak. A cyst was isolated from the water supply during an extended outbreak that resulted in 359 confirmed cases over a 7-month period. Subsequent outbreaks have clearly established the role of water in Giardia transmission. From 1965 through 1984, 90 waterborne outbreaks with 23,776 cases of giardiasis were reported in the United States. This report discusses the etiologic agent and examines drinking water outbreak occurrence and control.

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group

18. DISTRIBUTION STATEMENT	19. SECURITY CLASS *(This Report)*	21. NO. OF PAGES
RELEASE TO PUBLIC	UNCLASSIFIED	15
	20. SECURITY CLASS *(This page)*	22. PRICE
	UNCLASSIFIED	

EPA Form 2220-1 (Rev. 4-77) PREVIOUS EDITION IS OBSOLETE

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DRINKING WATER TRANSMISSION OF GIARDIASIS IN THE UNITED STATES

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ABSTRACT

For about a century after Giardia was named by Lambl in 1859, the occurrence of the organism in humans was not widely considered significant in explaining the etiology of disease. This organism is now recognized as a cause of disease that ranges from mild to severe and debilitating gastroenteritis. Beginning in 1965, drinking water became increasingly implicated as an important route of transmission. An outbreak occurred in a Colorado ski resort in that year. A survey of visitors to the resort revealed that 123 persons experienced acute enteritis, possibly giardiasis. Sewage contamination of the well water supply was thought to be the source of the organism.

Further evidence for waterborne transmission of Giardia came in 1975 with the first isolation of the organism from a water supply implicated in an outbreak. A cyst was isolated from the water supply during an extended outbreak that resulted in 359 confirmed cases over a 7-month period. Subsequent outbreaks have clearly established the role of water in Giardia transmission. From 1965 through 1984, 90 waterborne outbreaks with 23,776 cases of giardiasis were reported in the United States. This report discusses the etiologic agent and examines drinking water outbreak occurrence and control.

KEYWORDS

Giardia; giardiasis; waterborne outbreaks; drinking water; enteric disease.

INTRODUCTION

Giardia, a flagellated protozoan, was discovered in 1681 by Leeuwenhoek in a specimen of his own stool. It was rediscovered in 1859 by Lambl after whom the human species was named, Giardia lamblia. Even though Leeuwenhoek associated the presence of this organism with the diarrheal nature of his stool, the medical community over the years has not consistently recognized

the pathogenic potential of Giardia infection. Papers published in the medical literature during the first half of the 20th century report both findings of a strong association of giardiasis with gastroenteritis and the findings that the organism appeared to be a harmless invader of the gastrointestinal tract without health significance (Hartman and Kyser, 1941).

In 1937 a satisfactory treatment for giardiasis became available for the first time. The antimalarial drug quinacrine was found to eradicate the parasite from the stool of infected individuals resulting in relief from enteritis symptoms. This indirect evidence of the pathogenicity of Giardia increasingly led to a greater awareness of its public health significance. It had earlier been recognized that children had a higher incidence of infection. By the late 1950's, the pathogenic nature of giardiasis was considered serious enough by many pediatricians to advise treatment for all infected children. The organism is now recognized in the United States to be a cause of gastroenteritis, ranging from mild to severe and debilitating disease in all age groups. Although it is thought to be transmitted primarily by direct contact, the occurrence of water transmission has been clearly established.

THE ORGANISM AND THE DISEASE

The organisms in the genus Giardia have a worldwide distribution and infect the intestinal tract of a wide variety of vertebrates. The nomenclature for the organism that infects each host is confused because non-ambiguous criteria have not been established for Giardia speciation. Isolates from various animal hosts cannot be distinguished morphologically by microscopic examination. The species infecting humans (identified in the literature as G. lamblia, G. intestinalis or G. duodenalis) has been reported to also infect a number of wild and domestic animals (Levine, 1985). However, cross-species transmission of G. lamblia has not been unequivocally proven (Woo, 1984). Perhaps the strongest evidence is laboratory data for man-to-dog transmission and waterborne outbreak data for beaver-to-man transmission. It is inconclusive whether Giardia species common to other animals are infectious for man.

The organism exists in a trophozoite and a cyst form. The life cycle begins when an ingested cyst passes into the stomach of an exposed person where the excystation process begins. The resultant trophozoite resides in the upper small intestine and attaches to the epithelium by an adhesive disk. Here the trophozoites multiply by binary fission to large numbers and move down the intestinal tract. At some point in this downward movement the encystation process is begun and most of the organisms exit the infected host in the cyst form. Typical G. lamblia cysts are microscopically identified in stool specimens by their ovoid shape, size (~ 8 x 12 μm), and characteristic internal morphology (2-4 nuclei, axonemes and median bodies). Cysts are relatively resistant to inactivation under environmental conditions. Studies using dye exclusion and excystation capability as determinants of viability found cysts to survive for 24 days storage at 21 C and 77 days at 8 C. However, freezing and thawing cycles were destructive to the cysts and none were found viable after bringing a water suspension to the boiling point (Bingham et al., 1979).

The pathogenic mechanisms of Giardia infection are not well understood. Invasion of tissue of the intestinal tract by trophozoites has been reported. However, this may be uncommon as evidenced by the lack of a detectable humoral antibody response in most infected persons studied. Undoubtedly the attachment of the trophozoite to the intestinal wall by the adhesive disk could cause significant distress in massive infection. The predominant clinical feature is diarrhea, occurring in over 50% of those manifesting symptoms of giardiasis. Abdominal cramps, nausea and fatigue are common while vomiting and fever are symptoms rarely associated with giardiasis (Wolfe, 1984).

Asymptomatic giardiasis is likely to be the outcome of Giardia cyst ingestion. Only a few viable cysts may be needed to establish infection (Rendtorff, 1979). However, pathogenic giardiasis appears to be increasing. Major waterborne outbreaks have occurred in recent years with very high symptomatic attack rates in the exposed populations. The medical community is becoming increasingly aware of Giardia infection as a cause of gastrointestinal problems in their patients (Sealy & Schuman, 1981). If this had been the case in earlier years, it is unlikely that the controversy over the pathogenicity of this organism would have developed. Whether the perceived increased disease occurrence is largely due to improved physician recognition or involves important organism or host factors is unclear. Studies on differences in strain virulence of the organism over the years have not been conducted nor does information exist on the importance of significant host factors.

WATERBORNE OUTBREAKS

The first reported U.S. waterborne outbreak of giardiasis occurred in 1965 at a Colorado ski resort (Moore et al., 1969). At least 123 vacationers experienced prolonged diarrhea after ingesting water at the resort. The intestinal illness was rather severe with abdominal cramps and weight loss reported by a large number of the infected persons. Stool specimens were examined from 59 persons and Giardia cysts were isolated from 42%. Enteric bacterial and viral tests did not reveal an etiological agent. Subsequent to the outbreak, sewage from a leaking pipe that contaminated the ground water supplying the resort was found to contain Giardia cysts. These findings were consistent with the hypothesis of a waterborne Giardia outbreak even though the agent was not recovered from the drinking water.

It was almost 10 years later in 1974 before an outbreak occurred from which Giardia was actually isolated from an incriminated water supply. Over a 7-month period, 359 cases of giardiasis with vomiting and diarrhea occurred in a northeastern city of about 50,000 population. The surface water reservoir was suspected as the source of the cysts. Concentrates from water samples taken at the treatment plant intake were fed to pathogen-free beagle puppies. Two of 10 exposed pups developed giardiasis. Microscopic examination of water sediments from the treatment plant also revealed a Giardia cyst. The drinking water source could have been contaminated with untreated human wastes and this was considered the likely source of the organisms causing the outbreak (Shaw et al., 1977).

The reporting of waterborne outbreaks by state and local health units to a central health authority is not required in the U.S. However, a voluntary program of reporting and assistance in outbreak investigations, administered by the U.S. Public Health Service with assistance from the U.S. Environmental Protection Agency, has been in place for many years. Table 1 shows the giardiasis outbreak record obtained from this program since the first reported outbreak in 1965. The 1985 data are not yet available. The number of reported outbreaks has increased significantly from the 5-year period 1965-1969 to 1980-1984. This most assuredly is partially due to the increased awareness of Giardia as a waterborne pathogen and improved cyst detection and identification procedures. However, it is believed by many that the actual occurrence of waterborne giardiasis is on the increase.

TABLE 1 Waterborne Outbreaks of Giardiasis in the United States*

Period	Outbreaks	Cases
1965-1969	2	142
1970-1974	13	5,161
1975-1979	19	12,695
1980-1984	56	5,778
Total	90	23,776

*from Craun and Jakubowski, 1986

It is interesting to note that the significant increase in outbreaks for 1980-84 over the period 1975-79 caused 46% fewer cases. This difference reflects three large outbreaks in the years 1978 and 1979. The large 1978 outbreak in a Colorado ski community caused an estimated 5,000 cases of giardiasis. A surface stream supplying water that was minimally disinfected before distribution received a large volume of raw sewage as a result of a sewer line break. This was believed to be the source of the organism and the low disinfection level was apparently unable to inactivate cysts in the drinking water (CDC, 1978).

A total of about 5500 cases of giardiasis occurred in two waterborne outbreaks in 1979. Between September and December 1979, an estimated 3500 cases of giardiasis were reported in residents of Bradford, PA (Lippy, 1981; Craun, 1984). The surface water supply consisted of three impoundments and chlorination was the only treatment provided. Residents complained of muddy water due to heavy rains prior to the outbreak (turbidities in excess of 10 NTU were recorded on numerous occasions). Chlorination was not continuous and was ineffectual due to antiquated equipment and improper procedures for determining the applied dosage necessary to produce a residual in the distribution system. Coliforms were detected in the water prior to and during the outbreak but these findings only led to the collection of check samples and not to corrective action. Giardia cysts were detected in water samples and in a beaver living on the watershed.

The second largest outbreak in 1979 was attributed to direct cross-connections between sewage effluent used for irrigation and the potable water system of a shallow well supply. About 2000 cases of giardiasis occurred in visitors to a state park in Lake Havasu City, AZ (Lippy, 1981; Craun,

1984). Many of the affected individuals were from different health jurisdictions and the outbreak was recognized only because visitors to the campground reported their illness to local authorities who then contacted the state agencies. A multi state investigation then revealed the common source exposure and resultant disease outbreak.

A relatively small but significant outbreak is included in the 1984 data. In a Pennsylvania city of 50,000 population, 342 confirmed giardiasis cases occurred over a few months time in a pattern indicative of a common source outbreak. Giardia cysts were isolated from the source water, a river that receives upstream sewage discharge. The waterborne nature of this outbreak was atypical in that it was associated with drinking water that had been subjected to full conventional treatment. Even though it routinely met standard water quality parameters, the water treatment plant was about 80 years old and had operational deficiencies in the filtration process. The peak incidence of cases occurred within the typical giardiasis incubation period of an event recorded in the water utility's records (Table 2).

TABLE 2 Weekly Averages for Water Quality Parameters During Period of Waterborne Giardia Outbreak in a Pennsylvania Community

Date (Wk. Ending)		Turbidity (NTU)		Chlorine (mg/L)		pH	Coliforms
		Raw	Finished	Free	Total		Pos. Tubes/Inoc. Tubes
Dec.	3	34	0.24	1.1	1.3	8.5	1/35
	10	40	0.28	1.0	1.2	8.6	1/35
	17	24	0.37	1.0	1.2	8.6	0/35
	24	15	2.80	1.0	1.3	8.0	1/35
Jan.	7	9	2.70	1.3	1.6	8.8	0/35
	14	5	0.90	1.3	1.5	8.4	1/35
	21	5	0.52	1.2	1.5	8.5	2/35
	28	34	0.65	1.2	1.4	8.5	2/35
Feb.	4	15	0.49	0.9	1.2	8.6	2/35

During a period in late December 1983 and early January 1984, an unusually high demand for water left insufficient volume for backwashing the filters. The finished water turbidity increased from a weekly average of <1.0 to 2.8 turbidity units. Disinfectant residual levels and coliform indicator levels were well within normal limits (Akin and Hoff, 1985).

Research data mentioned later in this report have shown that minor increases in the turbidity of filtered water can result in the breakthrough of particles in the size range of Giardia cysts. It is very tempting to speculate that sudden relatively minor shifts in the quality of water produced from a source with significant Giardia contamination was the cause of this outbreak. At the alkaline pH of the water, the form and level of chlorine residual appeared to be inadequate to inactivate cysts that penetrated the rapid sand filters. Subsequent sampling of the source water at the plant

intake by Pennsylvania authorities has revealed consistent Giardia presence. Sampling of the finished water yielded an occasional positive finding. Data relative to this outbreak indicate that currently accepted water quality and treatment criteria may be inadequate to prevent Giardia transmission.

A major waterborne giardiasis outbreak occurred in late 1985 and is still under investigation (G.P. Kent, personal communication). In a northeastern city of about 50,000 residents, it is estimated that up to 8300 persons may have been infected. If so, this would be the largest reported Giardia outbreak in the U.S. Epidemiological investigation showed a typical common-source epidemic curve and implicated drinking water as the exposure route. Almost all cases had diarrhea with a mean duration of 12 days.

The outbreak coincided with the addition of a third surface water source to the community supply. Giardia cysts infectious to a laboratory animal model were recovered from the added source. The attack rate for giardiasis in the section of the city served exclusively from the added source was 4 to 5 times higher than sections served by the other two water sources. There was additional circumstantial evidence that implicated water from the added source as the origin of the outbreak. An increase in turbidity of the water supply was associated with the change in water distribution brought about by bringing the additional source on line. Also, problems with chlorination equipment resulted in reduced levels of chlorine (to undetectable levels) in water derived from the added source. Additional treatment and water quality information relative to the outbreak period has not been made available.

There was no evidence of sewage contamination in any of the three water sources. However, evidence of recent beaver activity was apparent. One stool specimen from nine beavers trapped near the added source was positive for Giardia.

SOURCES AND OCCURRENCE OF GIARDIA IN WATERWAYS

Giardia organisms can infect man and about 40 other species of animals (Kulda and Nohynkova, 1978). Cysts can therefore enter waterways through fecal or sewage discharges of man or through the droppings of a variety of domestic and wild animals. Beavers have been implicated in several waterborne outbreaks. However, proving that they were the source of contamination has not been possible because there is no way to identify the species of origin of a cyst detected in a water sample. Because of their aquatic habits, beaver and muskrat have a high potential for contaminating waterways. A survey throughout Washington State over a three-year period found the prevalence of Giardia infection in beavers to range from 6 to 19%, and in muskrats, from 35 to 43% (Frost et al., 1980). Another survey, confined to central Washington State, found about 83% of muskrats infected with Giardia (Pacha et al., 1985). The prevalence rates for Giardia infection in beaver and muskrat are currently being determined in watersheds located in one midwestern and four northeastern states. Preliminary results indicate that significant percentages of animals are infected in all five states (unpublished data).

In pristine areas with little or no human population, animals would likely be the primary source of waterway contamination. Cyst densities have been calculated for several streams in Washington State using beaver population size on the watershed, Giardia prevalence rate, stool size, number of cysts per gram of stool and stream flow rate (Jakubowski, 1984). Estimated cyst densities ranged from 1.4×10^{-6} to 9.3×10^{-6}/gal (3.8 L) in six streams during conditions of maximum flow. However, during conditions of minimum flow, estimated cyst densities ranged from 1.3 to 245/gal (3.8 L) in four streams. Although there is considerable uncertainty in the data for many of the factors used in these calculations, the results suggested that a few infected animals could heavily contaminate a small water system.

Estimates of cyst densities in sewage have also been prepared taking into consideration cyst shedding rates in humans, stool size, per capita water usage rates in the U.S. and percentage of the population that might be infected with Giardia (Jakubowski, 1984). These calculations suggested that the density of cysts in raw sewage could range from 9,600 to 240,000 cysts/L if 1 to 25% of the population, respectively, were infected. Until recently, there has been few data on the actual densities of cysts in sewage. Sykora (1986) has examined raw and treated sewage from several treatment plants. The densities of cysts in raw sewage ranged from 7 to 1242/L. The efficiency of the method used to detect and enumerate cysts ranged from 6 to 28%, so the actual cyst densities may be up to 16 times higher than those found. The cyst density may also have been underestimated because strict criteria were used in the identification and enumeration of cysts (Sykora, personal communication).

Giardia are not free-living protozoa, and they are not likely to reproduce outside of a host animal. Since they would probably be present at low densities in water supplies, concentration of samples is necessary for detection of cysts. A cultural technique is not available and current methods involve microscopic examination of large-volume concentrates for detection and identification of cysts. Procedures used in our laboratory involve a modification of a tentative or reference method developed by a consensus panel in 1980 (Jakubowski, 1984 and 1984a; APHA, 1985). Briefly, at least 100 gal (380 L) of raw or finished water is passed through a 1 µm nominal porosity depth filter. The retained particles are extracted by washing the filter fibers and then concentrated by sedimentation or centrifugation. The Giardia cysts are separated as much as possible from other particles in the sample by isopycnic centrifugation using sucrose or Percoll gradients. The purified concentrate may be stained with iodine and examined by light microscopy. Alternatively, an indirect fluorescent antibody (IFA) staining technique is available (Sauch, 1985), using fluorescence to aid in detecting cysts and phase microscopy to confirm identification. With either the iodine or the IFA techniques, presumptive identification of Giardia cysts is made by size and shape. Observation of at least two internal morphological characteristics (nuclei, axonemes, median bodies) confirms their identification.

Giardia methods are in a state of flux and there is a lack of supporting data for many of the procedures used (Jakubowski et al., 1984). Little or no published information is available on the efficiency, precision and sensitivity of the methods with different waters. Consequently, quantita-

tive data on cyst densities in water should be interpreted with caution, even when the data are from a single laboratory. Examples of cyst densities occurring in water at various locations in the U.S. are shown in Table 3.

TABLE 3 Giardia Cyst Densities in Water Supplies

State	No. of Samples	% Positive	Mean No. of Cysts/ 380 L (Range)
Oregon*	40	28	1 (0.1-5)
Idaho*	53	47	2 (0.05-7)
Wyoming**	147	14	23 (0.6-324)
Pennsylvania***	128	27	113 (0.2-680)
Totals	368	24	48 (0.05-680)

*Data provided by G.J. Vasconcelos (personal communication)
**Willey et al., 1986
***Data provided by W.P. Bancroft (personal communication)

The data shown in Table 3 were produced by three different laboratories and include raw and treated water samples. The Oregon and Idaho results are from a variety of supplies in those states; the Wyoming data are from 80 supplies throughout the state and the Pennsylvania data are from a single surface water supply. Almost all of the Pennsylvania positive samples were from the raw water, which is a river supply subject to sewage effluent discharges upstream. The samples from the other states include pristine and populated areas. The maximum density of cysts reported was 1.8/L in the Pennsylvania supply. It should be realized that the microscopic detection of cysts in a sample does not distinguish between viable and non-viable cysts. Cysts that have been inactivated may be detected and identified along with viable cysts. Considering the wide distribution of Giardia infections in man and animals, it is likely that all surface waters are subject to contamination with cysts at some point in time. The public health significance of water contamination will depend on the density of cysts, the source of cysts relative to their infectivity for man, the survival of the organisms in the environment and the adequacy of water treatment.

WATER TREATMENT AND CONTROL

In the U.S., it has been generally believed that drinking water derived from contaminated surface water sources should be filtered and chlorinated to a residual of 0.5 to 1.0 mg/L for a contact period of at least 30 minutes to assure microbiological protection. Giardia outbreaks are often associated with water supplies derived from surface streams thought to be pollution free and that are therefore minimally treated with chlorine. This observation and the absence of definitive data has stimulated laboratory studies with water disinfectants to determine their effectiveness for Giardia cyst inactivation as determined by excystation capability.

In a study with Giardia lamblia conducted by Jarroll et al., (1981), four factors were shown to be important for cyst inactivation: chlorine concen-

tration, water temperature, pH and contact time. The study was designed to observe about 99.9% loss in excystation capacity as determined by microscopically evaluating at least 1000 cysts at each trial. The findings are summarized as follows: (1) at a temperature of 25 C, exposure to 1.5 mg/L for 10 minutes at pH values of 6, 7 and 8 inactivated all cysts; (2) at 15 C, exposure to 2.5 mg/L of chlorine inactivated all cysts in 10 minutes at pH 6 but some survived longer than 30 minutes at the alkaline pH values; (3) at 5 C, 1 mg/L of chlorine failed to kill all cysts at any of the three pH levels in 60 minutes; 2 mg/L produced total inactivation at pH 6 and 7 but not at pH 8 in 60 minutes.

Similar studies of Giardia lamblia inactivation have been conducted with ozone disinfection (Wickramanayake et al., 1984). At 25 C and 0.034 mg/L of ozone, the lowest concentration tested, all cysts were inactivated within 6 minutes. At 5 C, an ozone concentration of 0.11 mg/L was required to achieve similar inactivation. Ultraviolet (UV) irradiation has been used as an alternative to chemical disinfection of drinking water. Studies conducted by USEPA have found UV to be an ineffective germicide under practical usage for Giardia lamblia (Rice and Hoff, 1981).

Studies of the mouse species, Giardia muris, as a model for G. lamblia have been conducted with chlorine dioxide (Leahy, 1985). Experiments were conducted at pH 5, 7 and 9 at temperatures of 5 C and 25 C. Chlorine dioxide was very effective in cyst inactivation and, in contrast to chlorine, was increasingly effective as the pH was raised from 5 to 9. Like chlorine, it was more effective at the higher temperature. In comparative experiments, chlorine dioxide was about one order of magnitude more effective than free chlorine at 25 C and about two orders more effective at pH 9.

The relative resistance of Giardia cysts, compared to enteric bacteria and viruses, to disinfectants at levels typically used in water treatment has indicated the need for an additional treatment barrier. The USEPA is considering a requirement of mandatory filtration of all surface water sources as a needed barrier against Giardia and other contaminants of health importance. Studies on water filtration for Giardia removal have indicated that rapid sand, slow sand, and diatomaceous earth media can be effective barriers (Hendricks, et al., 1985). A recent pilot plant study using a mono media anthracite filter obtained from a treatment plant implicated in a giardiasis outbreak and alum as a flocculent showed that the filter medium was inadequate in particle removal (Logsdon, et al., 1985). This study provides a warning that filtration per se may not assure Giardia removal and that proper operation and evaluation of the process is essential for effective filtration.

These studies and others not cited here have shown that filtration can provide a very effective barrier to waterborne cyst transmission if the water is properly pretreated. However to do so, the process must be consistently operated at a level of control that is atypical in many plants. Sudden fluctuations in turbidity even within the allowed limits may result in cyst breakthrough.

CONCLUSIONS

Giardia lamblia has become recognized as a major cause of gastroenteritis in the U.S. Since 1971, it has become the most commonly identified pathogen in waterborne outbreaks. In addition to numerous small outbreaks, four have occurred in recent years with over 2,000 cases of giardiasis per outbreak. Reporting of outbreaks is voluntary and the record is most assuredly incomplete. Nonetheless, 90 outbreaks and 23,776 cases have been reported through 1984. Over half of these (56 outbreaks) occurred in the most recent 5-year reporting period. Whether these data reflect an increased awareness and identification or an actual increase in occurrence is unclear. They probably represent a combination of the two possibilites.

It is intriguing to speculate that a more virulent strain of *Giardia* has appeared in the U.S. either as a result of importation with humans or as a result of cross-infectivity in other animal hosts. The authors are not aware of data or studies to evaluate this speculation. Outbreak data have clearly shown that human fecal contamination of drinking water sources is not the only origin of infectious agents that may cause waterborne disease. Even though Koch's postulates have not been satisfied for cross-species *Giardia* infection, the epidemiological data are convincing that the *Giardia* species that infect beaver can cause pathogenic manifestations in man. Cysts excreted by a number of additional animal species that inhabit many watersheds are morphologically indistinguishable from *G. lamblia* and may be infectious for man. Therefore, adequate barriers against this organism must be included in the treatment regime for surface water sources.

Filtration with adequate chemical pretreatment is increasingly recognized as an important barrier to *Giardia* control. However, the process must be properly operated to be effective. In addition, it is recognized that adequate disinfection is essential in the prevention of waterborne giardiasis. Continued surveillance of giardiasis cases and outbreak occurrence as an integral part of water treatment evaluation is important in defining treatment criteria that will eliminate this waterborne hazard and assure confidence in the safety of a water supply.

ACKNOWLEDGEMENTS

The authors thank John Stoecker for providing the Pennsylvania data.

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